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Mathematical Analysis of Extremity Immersion Cooling for Brain Temperature Management*

XIAOJIANG XU, WILLIAM SANTEE, LARRY BERGLUND, and RICHARD GONZALEZ

Summary. Due to the low heat conductivity of body tissue, head surface cooling methods for management of the brain temperature during medical treatments often have limited utility. As blood flow rates and surface-to-volume ratios are generally high in the extremities, heat exchange between the body and the environment through the extremities is an important path for heat exchange. This study examines the effects of cold-water extremity immersion on brain temperature by simulation modeling. The work is based on a sixcylinder thermoregulatory model that predicts human thermoregulatory responses to heat, cold, and water immersion. An arteriovenous anastomosis (AVA) response algorithm was added to the base model. Arteriovenous anastomoses are assumed to be controlled by a combination of core and skin temperatures. Our series of simulation scenarios consists of resting in a hot environment (40°C, 75% relative humidity) until the brain temperature rises to 39°C, then continuing to rest for 1h under one of the following treatments: (A) no cooling; (B) hands immersed in 10°C water; (C) feet immersed in 10°C water; (D) hands/feet immersed in 10°C water. The simulation results indicate that within the first 30 min, the hands, feet, or hands/feet immersion cooling resulted in brain temperature drops of 1.7°C, 2.4°C, and 3.3°C, respectively, which correspond to cooling rates of 0.03°C/min, 0.04°C/min, and 0.05°C/min. The predicted values show that extremity immersion cooling is a viable mechanism for simple and effective control of brain temperature.

Key words. Brain cooling, Extremity immersion, Arteriovenous anastomosis cooling, Simulation, Modeling

Introduction

Due to the low heat conductivity of the body tissue, surface cooling methods for management of the brain temperature during medical treatments, e.g., helmet cooling, nasopharyngeal cooling, and partial immersion in cold water, often have limited utility and result in

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a high temperature gradient from the center to the periphery across the brain. A more effective mechanism of heat exchange is convection by blood. Since blood flow rates in the skin of extremities are high when vasodilatation occurs and arteriovenous anastomoses (AVAs) are open, the extremities become important sites for heat exchange between the body and the environment [1,2]. In this study, a model was used to evaluate the effects of cold water extremity immersion on the brain temperature response.

Methods

The thermoregulation of the body was analyzed by isolating the control mechanisms, i.e., active systems, from passive systems that are regulated by a control system, i.e., the system components. The elements of the body are described and defined in the model by dimensions, mass, thermal properties, and energy transfer mechanisms. In the model, the body is represented by six cylinders which are head, trunk, arms, hands, legs, and feet. Each cylinder consists of core, muscle, fat, and skin layers. Body temperature is regulated by the control system through thermoregulatory mechanisms of sweating, shivering, and variable blood flow rate. The core, muscle, and skin layer temperatures are weighted and summed to form an integrated body temperature. The afferent signal for the controlling system is the difference between the integrated body temperature and its threshold. Details of this thermoregulatory model have been described in a previous report [3].

This model was augmented with an AVA response algorithm of the extremities developed by Takemori and Shoji [4] which postulates that the AVAs are controlled by both core and skin temperature. The magnitude of hand or foot AVA restriction, on a 0–1 scale, is calculated from the deviations from the set points for core and mean skin temperatures. When the AVAs are completely open, the magnitude equals 1, and the AVA blood flow is set at a maximum value of 30 ml/min per 100 ml tissue. When AVAs are fully closed, the magnitude is 0, and the AVA blood flow is zero. Thus, the magnitude of AVA restriction can be calculated by:

$$O_{\text{hands}} = 0.148 \cdot (T_{sk} - 34.0) + 0.532 \cdot (T_c - 36.8) + 0.51 \tag{1}$$

$$O_{\text{feet}} = 0.148 \cdot (T_{sk} - 32.6) + 0.532 \cdot (T_c - 36.6) + 0.51 \tag{2}$$

where O_{hands} and O_{feet} are the magnitudes of AVA restriction for hands and feet, respectively, T_{sk} is the mean skin temperature (°C), and T_{c} is the core temperature (°C).

The simulation scenarios all begin with a subject exposed to a hot environment (40°C, 75% relative humidity) until the brain rises to 39°C. Then the subject rests for 60 min in one of the following conditions: (A) no cooling; (B) hands immersed in 10°C water; (C) feet immersed in 10°C water; (D) hands/feet immersed in 10°C water.

Results and Discussion

Predicted changes in the brain temperature with no cooling, and hands, feet, or hands/feet immersion in 10°C water are shown in Fig. 1. The brain temperature increases to slightly above 39°C when no cooling is applied. In contrast, it falls rapidly when the extremities are immersed in cold water. During the first 30 min of hands, feet, or hands/feet immersion, the brain temperature drops by 1.7°C, 2.4°C, and 3.3°C, respectively. The corresponding cooling rates are 0.03°C/min, 0.04°C/min, and 0.05°C/min. These predicted cooling rates are in reasonable agreement with reported cooling rates of about 0.04–0.06°C/min with hands or feet

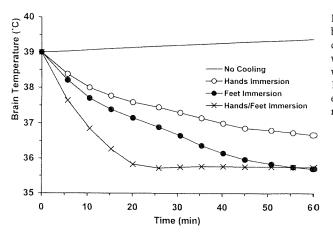


FIG. 1. Predicted changes in the brain temperature with no cooling, hands immersion in 10°C water, feet immersion in 10°C water, or hands/feet immersion in 10°C water when resting at an environment of 40°C and 75% relative humidity

immersion in 10°C water [5,6]. The potential effect of extremity cooling is also demonstrated by calorimeter studies that found the heat loss from hands or feet ranging from 100 to 150 W [5,7,8].

Blood flow rates in the extremities are critical components for predicting brain temperature responses. The maximum predicted blood flow in a hand or foot, including both vasodilatation and AVA opening, are 48 ml/min per 100 ml tissue and 45 ml/min per 100 ml tissue, respectively. These values are within range of the hand blood flows of about 70 ml/min per 100 ml tissue [9]. Digital blood flows can reach a maximum of 70–120 ml/min per 100 ml tissue [10]. Therefore, the prediction is reasonable and may even underestimate the cooling rates. Additional experimental studies are required to validate these rates.

The proposed mechanism for brain cooling due to cold water extremity immersion begins with a high rate of blood flow in the extremities due to vasodilatation and open AVAs. Blood temperature decreases as the blood flows through extremities immersed in cold water. The cold venous blood then returns to the heart and decreases the temperature of the central blood supply. The cooler arteriole blood flowing to the brain then results in brain cooling.

The surface-to-volume ratio of the extremities is about five times greater than the body as a whole. This facilitates the heat loss from the blood in the hands and feet to cold water. Hence, as long as there is high blood flow to the extremities, cold water immersion of the extremities can result in rapid brain cooling. An advantage of extremity cooling of the brain relative to more direct surface cooling of the head is that the temperature gradient from the center to the periphery is small.

Conclusion

Model prediction results show that extremity immersion cooling is a reasonable mechanism applicable for simple and effective means of controlling brain temperature. This model construct has applications for increasing our understanding of individual's physiological state.

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